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Issue Date: 13 April 2006

IN THE MATTER OF:

OLA MAE PRICE (widow of deceased miner William Price),
Claimant,

v. Case No.: 1997-BLA-1676

CONSOLIDATION COAL CO.,
Employer,

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-in-Interest.

DECISION AND ORDER ON REMAND AWARDING SURVIVOR'S BENEFITS

On June 28, 2005, the Benefits Review Board (Board) issued a *Decision and Order* vacating in part the June 16, 2004 *Decision and Order on Remand Awarding Survivor's Benefits*. The Board held that the opinions of Drs. Castle and Zaldivar must be re-weighed along with the opinions of Drs. Rasmussen, Buddington, Buono, and Ducatman to determine whether the miner's pulmonary impairment is related to his 42 year history of coal mine employment. Specifically, the Board directed as follows:

> [T]he administrative law judge must weigh the medical opinions of Drs. Castle, Zaldivar, Rasmussen, Buddington, Buono, and Ducatman and determine whether they are reasoned and documented and whether they contain adequate explanations of their conclusions as to whether this specific miner's pulmonary impairment was related to dust exposure in coal mine employment. Under the law of the United States Court of Appeals for the Fourth Circuit, the administrative law judge must address the qualifications of the physicians, the sophistication of their opinions, and the extent to which their conclusions are supported by the underlying objective evidence. (citations omitted). If the administrative law judge finds that claimant has established the existence of pneumoconiosis after weighing all of the evidence relevant to Section 718.202(a)

together, he must then address the issue of death due to pneumoconiosis pursuant to Section 718.205(c).

Slip op. at 6-7.

On the other hand, the Board held that it was proper to discredit the opinions of Drs. Abernathy, Endres-Bercher, Kress, and Fino with regard to the existence of legal pneumoconiosis.[1] Moreover, it concluded that the undersigned Administrative Law Judge properly concluded that clinical pneumoconiosis was not established through the chest x-ray evidence at 20 C.F.R. § 718.202(a)(1).

I

Medical opinion evidence under 20 C.F.R. § 718.202(a)(4) (2004)

All of the examining and consultative physicians of record conclude that the miner suffered from severe, progressively worsening chronic lung disease. Drs. Rasmussen, Buddington, Ducatman, and Buono concluded that the miner's respiratory impairment was due, at least in part, to his coal dust exposure as well as to tobacco abuse. Drs. Castle and Zaldivar conclude that the miner did not suffer from coal workers' pneumoconiosis and his respiratory impairment was due to tobacco abuse. Some of these physicians also noted an "asthmatic component" to the miner's respiratory condition. Physical examinations of the miner consistently revealed the presence sounds in the lungs, including wheezing, distant breath sounds, or rhonchi.

A. Dr. James Castle

In his November 17, 1997 report, Dr. Castle stated that he observed no physical findings that would indicate the presence of coal workers' pneumoconiosis. He noted a history of smoking one and one-half packs of cigarettes per day for 45 years, which he later translated to a 50 to 60 pack year smoking history during his deposition. *Ex.* 6 at 13.[2] He further noted a coal mine employment history of 30 years underground and "about 10 years above ground."

Dr. Castle opined that radiographic evidence did not support a finding of pneumoconiosis. Pulmonary function testing demonstrated a moderately severe obstructive airways disease, which progressed from 1987 until the miner's death. In Dr. Castle's view, this

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[1] In a previous *Decision and Order* dated March 19, 2003, the Board held the following: (1) it was proper to accord little weight to the opinion of Dr. Morgan with regard to the existence of pneumoconiosis; (2) it was proper to credit the opinions of Drs. Ducatman, Buono, and Rasmussen that smoking and coal dust exposure "led to the miner's fatal respiratory failure"; (3) it was proper to accord less weight to the opinions of Drs. Fino, Castle, and Endres-Bercher regarding the cause of the miner's death; (4) Claimant established 42 years of coal mine employment and a 35 to 45 pack year smoking history; and (5) pneumoconiosis, if present, was caused by coal dust exposure under 20 C.F.R. § 718.203(a).

[2] Dr. Castle stated that he reviewed a questionnaire submitted by Claimant wherein she stated that the miner smoked one pack of cigarettes per day for 45 years, before quitting in 1985.

was consistent with tobacco smoke induced pulmonary emphysema and chronic bronchitis. Dr. Castle also noted that the miner suffered from gas trapping and a reduction in diffusing capacity, which were consistent with pulmonary emphysema.

Dr. Castle opined that "[w]hen coal workers' pneumoconiosis causes clinically significant impairment, it does so by causing a mixed irreversible obstructive and restrictive ventilatory process." Moreover, he stated that, if coal workers' pneumoconiosis was significant enough to cause gas trapping, then "very significant changes" above a Category 1 would be noted in the chest x-rays, which was not the case here.

Dr. Castle noted that the miner's blood gas study results indicated a "frank respiratory insufficiency" with an elevation in the PCO2. He noted that coal workers' pneumoconiosis causes persistent hypoxemia without causing a CO2 elevation. Dr. Castle concluded that the miner's medical data was "absolutely typical of respiratory failure associated with tobacco smoke induced pulmonary emphysema."

Dr. Castle testified, during his December 1997 deposition, that the miner had evidence of a "moderately severe degree of obstructive airways disease which (he) felt had progressed from about 1987 until his demise." *Ex.* 6 at 12. Dr. Castle found no coal workers' pneumoconiosis based on a majority of chest x-ray readings. *Ex.* 6 at 14. He noted the following:

> As might be expected in an individual with tobacco smoke-induced pulmonary emphysema, (the miner) developed a progressive respiratory insufficiency such that he had now developed progressive hypoxemia requiring oxygen therapy, and then the finding of what we call hypercarbia, or increased PCO2.
>
> That is – those are hallmarks of respiratory failure due to tobacco smoke-induced pulmonary emphysema, and are not associated with the findings of coal workers' pneumoconiosis.

Ex. 6 at 15-16. After noting that the miner last worked as a dispatcher and left the mines in 1985, Dr. Castle further stated the following:

> The hallmarks of this process are that it was progressive after he left the mines with no further coal dust exposure. He had progressive hypoxemia and CO2 retention, which is one of the hallmarks of respiratory failure from tobacco smoking.

Ex. 6 at 17. Dr. Castle maintained that the miner did not exhibit a mixed impairment, which further militated against a finding of coal workers' pneumoconiosis. *Ex.* 6 at 20.

Dr. Castle opined that the miner's "very, very significant smoking history" was "certainly enough exposure to cause him to develop severe obstructive pulmonary disease, and by that I mean bronchitis, emphysema, lung cancer, or vascular disease." *Ex.* 6 at 13. He recalled that Dr. Zaldivar's 1991 examination of the miner produced a carboxyhemoglobin level that "was

elevated well above normal" and indicated a continuing smoking habit. *Ex.* 6 at 13. The miner last worked in the mines in 1985, but was still smoking cigarettes in 1991. *Ex.* 6 at 15-16.

With regard to the asthmatic component of the miner's respiratory ailment, Dr. Castle testified that, when the disease had reached a "severe end of the spetrum," the miner's FEV1 improved by only 200 to 300 cc's and "[w]e may see a 200 to 300 cc improvement in the FEV1 in severe smokers that have airways disease related to their smoking." *Ex.* 6 at 18. Further, Dr. Castle stated that this "does not occur in people that have obstruction due to coal workers' pneumoconiosis." *Ex.* 6 at 18. Rather, the "obstruction in that circumstance is related to fibrosis around the areas where coal was deposited, and because of that fibrosis, it doesn't respond to a bronchodilator." *Ex.* 6 at 18-19.

Dr. Castle concluded that the miner suffered from panacinar emphysema as well as centrilobular emphysema, "which is the typical type of emphysema that is seen with people that are tobacco users or abusers." *Ex.* 6 at 21. Dr. Castle testified that neither form of emphysema is "associated with coal dust exposure in any way." *Ex.* 6 at 22. He concluded that the miner suffered from:

> . . . typical, what we would say almost garden-variety tobacco smoke-induced emphysema that followed a typical downhill progressive course, and he died in the same fashion that he would have had he never worked inside of a coal mine.

Ex. 6 at 22-23.

Dr. Castle is board-certified in internal medicine and pulmonary diseases. He is also a NIOSH certified B-reader. He is on the staff of Carilion Roanoke Memorial Hospital and Carilion Roanoke Community Hospital. Dr. Castle testified that his "practice is one of a consultative or referral-based practice in pulmonary diseases, and by that I mean I accept patients only on referral, and my practice consists of any and all types of pulmonary disease." *Ex.* 6 at 6. He sees "generally between ten and 14 or 15 patients in the hospital on a daily basis, and some of those people are critically ill on ventilators, and I will see between usually 14 to 18 patients in the office on a daily basis." *Ex.* 6 at 6. Dr. Castle also serves as a clinical professor of medicine at the University of Virginia School of Medicine. *Ex.* 6 at 7. He spends "probably 20 percent of (his) time doing clinical research in new drugs for patients with pulmonary disease." *Ex.* 6 at 7. Dr. Castle has not written articles specifically on pneumoconiosis, but he has been involved in research of chronic obstructive pulmonary disease, tobacco smoke-induced lung disease, emphysema, and lung cancer. *Ex.* 6 at 9.

B. Dr. George Zaldivar

Dr. Zaldivar agrees that the miner died due to severe obstructive lung disease. He concluded that this disease arose from tobacco abuse and asthma. Dr. Zaldivar noted that there was insufficient objective data to support a finding of coal workers' pneumoconiosis. He found the presence of a "severe irreversible airway obstruction with air trapping and a moderate diffusion impairment all of which were due to (the miner's) smoking habit."

After his November 22, 1991 report, which was based on examination and testing of the miner as well as a review of certain medical records, Dr. Zaldivar opined that the miner suffered from smoking-induced emphysema with an asthmatic component. He noted 40 years of coal mine employment as well as a smoking history of 45 pack years. Indeed, Dr. Zaldivar noted that the miner continued to smoke intermittently as the carboxyhemoglobin level during his April 23, 1991 examination of the miner "was high and compatible with the carboxyhemoglobin level of a smoker." On the other hand, Dr. Zaldivar noted that the carboxyhemoglobin level measured during Dr. Crisalli's April 12, 1991 examination was 2.0, which was "borderline between a smoker and a non-smoker."

Dr. Zaldivar found no evidence of coal workers' pneumoconiosis by chest x-ray. He further concluded that Dr. Rasmussen's opinion was incorrectly based on a finding of no air trapping; rather, Dr. Zaldivar stated that his testing revealed significant air trapping.[3]

Improvement of ventilatory function after use of a bronchodilator was, in Dr. Zaldivar's opinion, "significant only insofar as the percentage but not insofar as the absolute improvement." From this, he states that there was an "asthmatic component" to the miner's emphysema. Dr. Zaldivar states:

> In conclusion, my opinion is that Mr. Price suffers at this time from emphysema caused by smoking. Further, it is my opinion that Mr. Price is smoking intermittently at the present time. This accounts for the intermittently high carboxyhemoglobin level. Mr. Price has an asthmatic component to his obstruction but the main problem is emphysema caused by smoking.

Dr. Zaldivar issued a supplemental report on January 7, 1998 based on a review of certain medical records. Dr. Zaldivar noted some improvement on ventilatory testing after use of a bronchodilator attributable to an asthmatic component of the miner's disease. However, Dr. Zaldivar concluded that the miner also suffered from severe, irreversible airway obstruction with air trapping and a moderate diffusing impairment due to the miner's smoking habit.

Dr. Zaldivar reported that the miner continued to smoke at the time of his 1991 examination "as evidenced by the high carboxyhemoglobin level measured in (his) office." He found "no evidence of pneumoconiosis." He concluded that there was insufficient evidence to justify a diagnosis of coal workers' pneumoconiosis. Dr. Zaldivar opined that the miner suffered from a very significant pulmonary impairment due to smoking-induced emphysema. He questioned the possibility of the presence of asbestosis based on the chest x-ray conducted during his 1991 examination.

In this report, Dr. Zaldivar also noted disagreement with Dr. Rasmussen's citation of certain medical literature purporting to state that coal dust exposure may cause the development of centrilobular emphysema. Dr. Zaldivar states that this conclusion is incorrect and the literature cited by Dr. Rasmussen "is nothing more than a compendium of previous studies in the

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[3] Notably, in his January 20, 1987 report, Dr. Rasmussen found that the miner exhibited a "marked increase in dead space ventilation."

field of occupational pneumoconiosis and it does not state that centrilobular emphysema is a manifestation of coal workers' pneumoconiosis." Dr. Zaldivar further states the following:

> One major flaw of the papers referred to by Dr. Rasmussen is that the smoking habit of the coal miners which were said to have lost lung function due to coal mine work was inaccurately stated in the papers. The reason for this is that many of the papers related to coal mine work and the effect on lung function are based on the histories provided by the miners in the form of answers to questionnaires. The miners themselves were not examined and therefore a smoking or health history was not obtained.
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> Dr. Morgan accurately separated the pathological findings of centrilobular emphysema from that of focal emphysema. The two are easily distinguishable by the fact that the focal emphysema of coal workers' pneumoconiosis has a macule while centrilobular emphysema of smoking does not.

Dr. Zaldivar further noted, upon review of Dr. Fino's report, that the miner did not suffer from hyercarbia, meaning high carbon dioxide content in the blood stream, at the time the miner quit working; rather, the condition "occurred later in life as he continued to smoke and as he continued to destroy his lungs through emphysema caused by smoking."

Dr. Zaldivar concluded that there was insufficient objective evidence to support a diagnosis of coal workers' pneumoconiosis:

> There was a very significant pulmonary impairment present. None of the impairment was due to pneumoconiosis. All of the impairment was due to emphysema caused by his smoking habit. Mr. Price had the same clinical course of any individual who smoked as must as he did and who developed emphysema as a result of his smoking habit. As mentioned by Dr. Castle, had Mr. Price never worked in a coal mine, no one would have raised the issue of coal mine work possibly contributing to his pulmonary impairment because the impairment and death was fully explained by his smoking habit.

Further, Dr. Zaldivar opined:

> Even if Mr. Price were found to have early simple pneumoconiosis by tissue analysis of his lungs, my opinion regarding the cause of the severe pulmonary impairment which he had is that he had emphysema caused by smoking. Mr. Price had bullae radiographically. It is a manifestation of emphysema which causes centrilobular and in the case of a bullae, panacinar emphysema.

Dr. Zaldivar is board-certified in pulmonary disease, internal medicine, and sleep disorders. He is also a NIOSH certified B-reader. Dr. Zaldivar serves as a clinical professor of medicine at the West Virginia University School of Medicine. He also serves as the Director of Respiratory Therapy and Director of the Sleep Disorder Center at the Charleston Area Medical Center (CAMC). As far as medical staff positions, Dr. Zaldivar has been Chief of the

Department of Medicine at CAMC since 1987. He has also been a member of the Quality Assurance Committee and Executive Committee at the CAMC since 1985 and 1987, respectively. In 1990, he and Dr. W.K.C. Morgan had the article, "Blood Gas Analysis As a Determinant of Occupationally Related Disability," published in the Journal of Occupational Medicine.

C. Drs. Alan Ducatman and Gina Buono

Drs. Ducatman and Buono reviewed certain medical records and issued a joint report on October 21, 1997. They reported 35 to 40 years of coal mine employment as well as a 35 to 45 pack year history of smoking cigarettes, where the miner quit in 1986. They concluded that severe chronic obstructive pulmonary disease was the immediate cause of the miner's death and the "[c]auses are cigarette smoking (major cause) and coal workers' pneumoconiosis (contributory cause)." They found the presence of simple coal workers' pneumoconiosis based on a single positive chest x-ray interpretation by a B-reader. Drs. Ducatman and Buono further diagnosed the presence of right ventricular heart failure secondary to severe lung disease, and coronary artery disease caused by cor pulmonale and cigarette smoking. They concluded that the miner died due to respiratory disease arising from smoking and coal dust exposure.

These physicians noted the following with regard to Dr. Rasmussen's report and findings on examination:

> Mr. Price was noted in 1987 by Dr. Rasmussen to have had progressive effort dyspnea for the past 12-15 years. He had episodes of coughing, paroxysmal nocturnal dyspnea and ankle swelling. He was using Theodur to control his respiratory symptoms. On exam done by Dr. Rasmussen, Mr. Price had tachypnea, wheezing, and decreased breath sounds.
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> . . .
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> Ventilatory studies showed moderate restrictive disease and severe obstructive disease. Blood gases revealed reduced arterial oxygen and a diminished diffusing capacity. Dr. Rasmussen performed an exercise test which showed marked impairment in gas exchange with hypoxia, indicating severe pulmonary insufficiency.

Drs. Buono and Ducatman further noted that Dr. Rasmussen, in his August 1989 letter, attributed the miner's ventilatory insufficiency to coal dust exposure and smoking and "Dr. Rasmussen points out that pulmonary function tests of Mr. Price did not show an increase in lung capacity as might be expected with chronic obstructive lung disease." After reviewing the miner's terminal hospitalization records, Drs. Buono and Ducatman diagnosed severe chronic obstructive pulmonary disease as the immediate cause of death, of which the major cause was smoking and the contributing cause was coal dust exposure. Simple coal workers' pneumoconiosis was diagnosed on chest x-ray. The miner suffered from right ventricular heart failure secondary to severe lung disease. Finally, Drs. Buono and Ducatman also diagnosed coronary artery disease stemming from cor pulmonale and cigarette smoking.

Drs. Buono and Ducatman are professors of medicine at the University of West Virginia School of Medicine. One of the physicians is also Director of the Institute of Occupational and Environmental Health and Chair of the Department of Community Medicine. Dr. Buono has a Master's degree in public health. Dr. Ducatman has a Master's degree in science.

D. Dr. Richard S. Buddington

Dr. Buddington examined the miner and issued a report on April 19, 1985. He noted a 45 pack year history of smoking cigarettes and a 40 year history of coal mine employment. Based on pulmonary function and blood gas testing as well as physical examination, Dr. Buddington concluded that the miner suffered from a moderate, chronic respiratory impairment. He attributed the miner's respiratory impairment to coal dust exposure based on "many years of mining." He did not offer any further explanation of his diagnosis. After reviewing a positive x-ray reading by Dr. Bassali, Dr. Buddington reiterated his diagnosis of coal workers' pneumoconiosis in a letter dated February 20, 1987. Dr. Buddington stated that the miner complained of chronic productive cough that had been ongoing for 15 years. He further reported complaints of dyspnea with lifting or "any minor activity" as well as wheezing and paroxysmal nocturnal dyspnea. On examination, heart sounds were distant and the lungs were "clear to auscultation and percussion." Dr. Buddington opined:

> The patient has moderate chronic respiratory impairment based on history and physical and abnormal arterial blood gases at rest . . . and abnormal ventilatory testing. This degree of impairment indicates that the patient does not have dyspnea at rest and may have dyspnea during the usual activities of daily living. The patient may be able to perform some heavy physical labor for brief periods of time with long periods of rest in between.

Dr. Buddington diagnosed moderate chronic pulmonary disease due to "many years of mining exposure." He noted that the miner also had an abnormal resting EKG but, according to Dr. Buddington, "[a]ny possible cardiac disease did not influence (the) pulmonary findings."

Dr. Buddington is board-certified in anatomical and clinical pathology. He performed his internship in pathology at Duke University Medical Center. He currently serves as pathologist for the Intermountain Pathology Associates in Bristol, Tennessee. Dr. Buddington is also Associate Pathologist at Bristol Memorial Hospital and he serves as the Director of Laboratories at Johnson Memorial Hospital.

E. Dr. D.L. Rasmussen

Dr. Rasmussen examined and tested the miner and issued a report on January 20, 1987. He noted a 41 and one-half year history of coal mine employment, where the miner worked as a motorman, shuttle car operator, trackman, roof bolter, and dispatcher, as well as a 35 pack year cigarette smoking history, quitting in 1986. Dr. Rasmussen further reported complaints of "progressive effort dyspnea for 12 to 15 years" as well as a chronic productive cough, episodes of wheezing, orthopnea, paroxysmal nocturnal dyspnea, and ankle swelling. Examination of the

lungs revealed audible wheezes and rhonchi. The AP diameter of the chest was increased. Breath sounds were "markedly reduced." Ventilatory testing was of "relatively poor quality" but it indicated the presence of a "moderate restrictive and severe obstructive insufficiency." The maximum breathing capacity was "markedly reduced" and the miner's total lung capacity was at the lower limits of normal. Blood gas testing demonstrated "markedly" reduced resting values as well as a "markedly reduced" diffusing capacity. The miner's EKG was normal after a treadmill exercise. However, the heart rate was moderately excessive and there was a "very marked increase in ventilation, marked increase in dead space ventilation." Dr. Rasmussen determined that the studies revealed a "very severe pulmonary insufficiency." He concluded that the miner suffered from totally disabling respiratory disease due to tobacco abuse and coal dust exposure.

Based on his February 4, 1987 examination, Dr. Rasmussen diagnosed the presence of coal workers' pneumoconiosis based on a chest x-ray and prolonged exposure to coal dust. He concluded that the miner was totally disabled due to smoking and coal dust induced lung disease.

By supplemental report dated August 22, 1989, Dr. Rasmussen reviewed certain additional medical records. He reiterated that the miner suffered form occupational lung disease, which contributed to his overall totally disabling respiratory impairment. He stated the following:

> This patient's markedly reduced diffusing capacity and his abnormal gas exchange during exercise . . . coupled with the marked reduction in diffusing capacity clearly indicates that this patient has pulmonary parenchymal destruction. Coupling this with his severe ventilatory impairment would lead one to believe that he has pulmonary emphysema. Interestingly, however, he has no increase in total lung capacity suggesting that he has at least a significant component of pulmonary fibrosis. Both emphysema and fibrosis are consistent with occupational pneumoconiosis.

Although the miner demonstrated some bronchoreversibility on ventilatory testing, Dr. Rasmussen was not convinced that a definitive diagnosis of asthma could be made. Moreover, he concluded that, even if the miner suffered from asthma, it "would be grossly insufficient to explain the degree of disability this patient has." As an example, Dr. Rasmussen stated that the miner's "marked reduction in diffusing capacity alone is indicative of totally disabling respiratory insufficiency." Moreover, a "reduced diffusing capacity is not characteristic of bronchial asthma" but it is "certainly . . . characteristic of both pulmonary emphysema and fibrosis." Dr. Rasmussen concluded that based on the miner's pattern of impairment, including his essentially normal lung volumes, his severe airway obstruction and his marked reduction in diffusing capacity, the miner suffers from occupational lung disease and this disease is "the major contributing factor to his totally disabling respiratory insufficiency."

In a supplemental report dated February 4, 1993, Dr. Rasmussen reviewed certain additional medical records and stated the following:

> There is general agreement that the patient has disabling pulmonary insufficiency. The principal physiologic abnormality appears to be that of severe pulmonary emphysema.

Noting that the chest x-ray evidence produced conflicting interpretations, Dr. Rasmussen cited to a study of x-rays and autopsy evidence available on 100 miners. The study revealed that 20 percent with severe macular pneumoconiosis, 30 percent with a moderate grade of macular pneumoconiosis, and 29 percent of individuals with micronodular pneumoconiosis had x-rays interpreted by three B-readers as negative for the presence of the disease. Dr. Rasmussen posits that a negative chest x-ray interpretation did not preclude the presence of pneumoconiosis in the miner's lungs. Moreover, Dr. Rasmussen stated that Drs. Zaldivar and Crisalli "ignored" a growing body of medical evidence that chronic obstructive pulmonary disease and emphysema can be caused by coal dust exposure, even in the absence of chest x-ray evidence. Indeed, he maintains that centrilobular and focal emphysema can arise from coal dust exposure and that the miner's emphysema was due, at least in part, to his coal dust induced lung disease. Again, Dr. Rasmussen concludes that the miner suffered from a totally disabling respiratory impairment due to coal workers' pneumoconiosis.

The miner died on January 8, 1997 as a consequence of respiratory failure and, in his September 22, 1997 report, Dr. Rasmussen reviewed additional medical records, including the miner's terminal hospitalization records, and he concluded that the miner developed a progressively severe respiratory insufficiency requiring multiple hospitalizations. Dr. Rasmussen emphasized that the miner "was employed for more than 40 years in the coal mining industry, much of which was prior to the institution of dust suppression in the coal mining industry" and "[m]ost of his work was at the face." Dr. Rasmussen opined that the miner's "respiratory failure can be attributed to his significant coal mine dust exposure, as well as a significant history of cigarette smoking." He noted that smoking is a "well-known cause of chronic obstructive pulmonary disease, including emphysema." However, Dr. Rasmussen cited to numerous medical articles and stated that "coal mine dust exposure is also known to be capable of producing chronic obstructive pulmonary disease, including emphysema." Dr. Rasmussen further stated that "death from chronic obstructive pulmonary disease is more prevalent among coal miners than in other occupational groups" and it is "noteworthy that this increased death is not specifically related to the appearance of the chest x-ray." He cited to articles and studies, including a September 1995 National Institute of Occupational Safety and Health (NIOSH) article titled "Criteria for a Recommended Standard Occupational Exposure to Respirable Coal Mine Dust," that smoking and coal dust exposure can cause centrilobular emphysema. Dr. Rasmussen concluded that coal workers' pneumoconiosis was a "material contributing factor" to the miner's death.

Dr. Rasmussen is board-certified in internal medicine and pulmonary diseases. He is also a NIOSH certified B-reader. He works for the Division of Pulmonary Medicine at the Southern West Virginia Clinic. He has held several special appointments directly related to the study of occupational pneumoconiosis, including the NIOSH Coal Mine Health Advisory Committee, NIOSH Mine Health Research Advisory Committee, National Advisory Committee of the United Mine Workers of America Coal Miners' Respiratory Clinic Program, West Virginia Workers' Compensation Advisory Board, NIOSH Ad Hoc Committee on Criteria for Disability

and Death by Coal Workers' Pneumoconiosis, and the U.S. Department of Labor's Committee for Disability Standards for the Federal Black Lung Compensation Program. Dr. Rasmussen has also been called to testify before both houses of the United States Congress as well as the West Virginia State Legislature on issues of occupational pneumoconiosis. In 1969, Dr. Rasmussen was awarded the American Public Health Association's Presidential Award for "exceptional service in the fight against black lung." Dr. Rasmussen has also authored a variety of published pieces, many of which address occupational pneumoconiosis and its effects.

Discussion and conclusions

Drs. Zaldivar, Castle, Ducatman, Buono, and Rasmussen[4] agree that the miner suffered from a totally disabling respiratory impairment. Indeed, Drs. Zaldivar, Castle, Ducatman, Buono, and Rasmussen agree that the miner had a moderately severe to severe obstructive lung disease. Although Dr. Rasmussen also found the presence of a restrictive impairment, he noted that the ventilatory testing was poor. Drs. Zaldivar, Castle, and Rasmussen also diagnose pulmonary emphysema and chronic bronchitis. The physicians agree that the miner's diffusion capacity was reduced. Drs. Castle and Zaldivar noted the presence of gas or air trapping and Dr. Rasmussen found a "marked increase in dead space ventilation" after exercise. Drs. Ducatman and Buono also found the presence of right ventricular heart failure secondary to the miner's severe lung disease as well as coronary artery disease due to cor pulmonale and smoking. Based on the foregoing, it is undisputed that the miner suffered from a multitude of respiratory conditions that left him disabled. Drs. Zaldivar, Castle, and Rasmussen, in particular, agree that the miner's severe obstructive lung disease and emphysema were totally disabling. Thus, the causes of these conditions will be examined.

Etiology of chronic obstructive pulmonary disease and emphysema

As previously noted, Drs. Zaldivar, Castle, Rasmussen, Buddington, Ducatman, and Buono, conclude that the miner suffered from chronic obstructive pulmonary disease. Drs. Rasmussen, Castle, and Zaldivar also diagnosed the presence of emphysema.[5] Drs. Zaldivar and

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[4] Dr. Buddington concluded that the miner suffered from moderate chronic respiratory impairment stemming from coal dust exposure and smoking at the time of his 1985 examination. He opined that the miner could perform heavy manual labor for intermittent periods of time with periods of rest in between.

[5] Drs. Zaldivar and Castle conclude that the miner had an asthmatic component to his emphysema. Dr. Rasmussen is not convinced that the miner had an asthmatic component to his respiratory impairment; rather, he states that it is possible that the miner exhibited increased cooperation after use of the bronchodilator. However, he notes that, if the miner did suffer from some form of asthma, this condition did not account for his residual, disabling impairment. In this vein, Dr. Rasmussen cites to the miner's "marked reduction in diffusing capacity" and states that this condition, standing alone, is "indicative of a totally disabling respiratory insufficiency." Further, Dr. Rasmussen notes that a reduced diffusing capacity is not characteristic of bronchial asthma, but it is characteristic of pulmonary emphysema and fibrosis. Dr. Zaldivar similarly states that improvement on ventilatory function after use of a bronchodilator was "significant only insofar as the percentage but not insofar as the absolute improvement." Although, Dr. Zaldivar diagnosed an "asthmatic component" to the miner's emphysema, he agreed that the miner suffered from a severe, irreversible obstructive pulmonary disease. Likewise, Dr. Castle noted that ventilatory testing revealed only 200 to 300 cc of improvement after use of a bronchodilator, which he maintains is seen "in severe smokers that have airways disease related to their smoking."

Castle attributed the miner's obstructive lung disease solely to his history of tobacco abuse, whereas Drs. Rasmussen, Buddington, Ducatman, and Buono concluded that the obstruction was caused by exposure to coal dust as well as smoking. With regard to the miner's emphysema, Drs. Zaldivar and Castle conclude that it is also only related to the miner's history of tobacco abuse. Dr. Rasmussen, on the other hand, concludes that it is related to coal dust exposure as well as smoking. Drs. Ducatman, Buono, and Buddington do not specifically address the presence or absence of emphysema.

A. Consideration of the Department's position, generally

In assessing the probative value of the physicians' opinions on this issue, it is proper to consider the Department's findings underlying promulgation of the amended regulations on December 20, 2000. *See* 65 Fed. Reg. 79920 (Dec. 20, 2000). Citation to the Department's medical findings underlying the amendments is proper for three reasons.

First, it is noted that the amended definition of "pneumoconiosis" at 20 C.F.R. § 718.201 (2004) is applicable to this claim pursuant to 20 C.F.R. § 718.2 (2004). *See also National Mining Ass'n. v. Dep't. of Labor*, 292 F.3d 849 (D.C. Cir. 2002). Consequently, medical findings contained in comments in support of the amended definition of pneumoconiosis at 20 C.F.R. § 718.201 (2004) are relevant.

Second, it is not unusual for courts to cite to, and consider, published comments underlying the promulgation of regulations in rendering their decisions. *See Mullins Coal Co. v. Director, OWCP*, 484 U.S. 135, 156 n. 29 (1988) (favorable discussion of the Department's comments underlying promulgation of 20 C.F.R. § 727.203(a) to determine that the agency did not intend that a single piece of qualifying evidence be sufficient to invoke the interim presumption); *Consolidation Coal Co. v. Director, OWCP [Stein]*, 294 F.3d 885, 892 (7th Cir. 2002) (favorable consideration of the Department's December 2000 comments with regard to use of CT-scans in assessing the presence or absence of pneumoconiosis); *Bonessa v. United States Steel Corp.*, 884 F.2d 726, 729 (3rd Cir. 1989) (favorable referral to the Department's 1983 comments to 20 C.F.R. § 718.205(c) in assessing causation). Therefore, consideration of the Department's findings as set forth in the comments to the amended regulations is proper. *Freeman United Coal Mining Co. v. Summers*, 272 F.3d 473 (7th Cir. 2001).

Third, according deference to the Department's medical findings after public rulemaking proceedings is proper. Indeed, the findings are based on an extensive public notice and comment rule-making period conducted in compliance with the strictures of the Administrative Procedures Act (APA). The comments are the product of the Department's extensive review of medical literature, studies, articles, and reports from a variety of medical experts and organizations, including the National Institute of Occupational Safety and Health (NIOSH), which is the congressionally mandated medical advisor for the Black Lung program. After considering divergent expert medical opinions from multiple sources, the agency made certain findings regarding the nature and characteristics of pneumoconiosis that are entitled to deference. As noted by the Seventh Circuit in *Stein*, it is proper to "defer to the Department of Labor's

reasonable judgment in resolving complex, technical issues that draw upon its familiarity and expertise with the diagnosis, prevention, and remediation of black lung disease." *Id.* at 892.

B. The physicians' opinions

In support of his opinion that the miner's respiratory impairments were not due to coal dust exposure, Dr. Castle reasons that the miner's ventilatory testing did not reveal a "mixed impairment," *i.e.* obstructive *and* restrictive characteristics, which militated against a finding that the condition was coal dust related. On the other hand, Dr. Rasmussen concluded that the miner's obstructive impairment was due, in part, to coal dust exposure. Drs. Buono, Ducatman, and Buddington offered opinions similar to that of Dr. Rasmussen. Moreover, while agreeing with Drs. Zaldivar and Castle that smoking is a "well-known cause of chronic obstructive pulmonary disease, including emphysema," Dr. Rasmussen also cited to numerous medical articles in support of a finding that coal dust exposure may cause solely obstructive impairments or emphysema as in this case.

Dr. Rasmussen's view is consistent with the Department's position. The Department has stated:

> Whether coal mine dust exposure can cause chronic obstructive pulmonary disease is a question of medical and scientific fact that will not vary from case to case; thus, it is an appropriate question for the Department to answer by regulation.

65 Fed. Reg. at 79938 (Dec. 20, 2000). According to the Department, chronic obstructive pulmonary disease is comprised of three disease processes—chronic bronchitis, emphysema, and asthma—which are present in this case. 65 Fed. Reg. 79939 (Dec. 20, 2000). The Department notes that "[e]ven in the absence of smoking, coal mine dust exposure is clearly associated with clinically significant airways obstruction and chronic bronchitis" and this "risk is additive with smoking." 65 Fed. Reg. at 79940 (Dec. 20, 2000). The Department further states that, while a claimant must demonstrate that the miner's obstructive lung disease arose out of coal dust exposure, medical opinions that exclude obstructive lung diseases from occupationally-related pathologies, are inconsistent with the Department's view**.** 65 Fed. Reg. at 79938 (Dec. 20, 2000). Thus, Dr. Rasmussen's opinion in this case is based on a premise that is better in accord with the Department's position, *i.e.* coal dust exposure may cause chronic obstructive pulmonary disease without any requirement that restriction also be present.

Drs. Castle and Zaldivar also diagnosed the presence of centrilobular and panacinar emphysema and posited that these conditions are "almost garden-variety tobacco smoke-induced." Dr. Castle cited to gas trapping and a reduced diffusing capacity as supporting a diagnosis of pulmonary emphysema. He testified that centrilobular and panacinar emphysema are not "associated with coal dust exposure in any way." Dr. Zaldivar agreed with Dr. Castle and expressed disagreement with Dr. Rasmussen's statement that coal dust exposure can cause the development of centrilobular emphysema. Dr. Zaldivar challenged the articles and studies cited by Dr. Rasmussen as containing inaccurate smoking histories of the miners who were studied. Dr. Zaldivar stated that centrilobular and focal emphysema are easily distinguishable as "the

focal emphysema of coal workers' pneumoconiosis has a macule while centrilobular emphysema of smoking does not."

In its comments, however, the Department noted that medical data supported a finding that "[c]entrilobular emphysema . . . was significantly more common among the coal workers." 65 Fed. Reg. at 79941 (Dec. 20, 2000). Indeed, the "severity of the emphysema was related to the amount of dust in the lungs" and "[t]hese findings held even after controlling for age and smoking habits." 65 Fed. Reg. at 79941 (Dec. 20, 2000). In one study, which involved pathological review of the lungs of 450 coal miners, the authors of the study found "emphysematous changes in 72% of miners who smoked, 65% of ex-smokers, and 42% of non-smoking miners . . .." 65 Fed. Reg. at 79942 (Dec. 20, 2000). Moreover, it was noted that 47% of "miners with no fibrotic lesions had emphysema." 65 Fed. Reg. at 79942 (Dec. 20, 2000).

Dr. Rasmussen concludes that the miner's chronic obstructive pulmonary impairment and emphysema were due to smoking and coal dust exposure. Moreover, Dr. Rasmussen states, similar to the opinion of Dr. Castle, that a reduction in diffusing capacity is "characteristic" of the presence of pulmonary emphysema. Dr. Rasmussen adds that the miner's reduced diffusing capacity is also characteristic of fibrosis. He states that these findings of pulmonary emphysema and fibrosis are consistent with a diagnosis of occupational pneumoconiosis. In particular, Dr. Rasmussen noted:

> This patient's markedly reduced diffusing capacity and his abnormal gas exchange during exercise . . . indicates that this patient has pulmonary parenchymal destruction. Coupling this with his severe ventilatory impairment would lead one to believe that he has pulmonary emphysema. Interestingly, however, he has no increase in total lung capacity suggesting that he has at least a significant component of pulmonary fibrosis. Both emphysema and fibrosis are consistent with occupational pneumoconiosis.

Dr. Rasmussen further opines that the miner's chronic obstructive pulmonary disease and centrilobular emphysema are caused by coal dust exposure as well as smoking. He persuasively explains that the miner's pattern of impairment in this case, including the essentially normal lung volumes, severe airway obstruction, and the marked reduction in diffusing capacity, supports a diagnosis of coal workers' pneumoconiosis and this disease "is the major contributing factor to (the miner's) totally disabling respiratory insufficiency" notwithstanding the negative x-ray interpretations of record. This is consistent with the Department's position that "coal dust exposure is associated with significant deficits in lung function in the absence of [clinical] CWP, reinforcing the view that COPD and CWP have independent risk factors." 65 Fed. Reg. at 79941 (Dec. 20, 2000). Moreover, the Department cites to medical literature as follows:

> [D]ust-induced emphysema and smoked-induced emphysema occur through similar mechanisms—namely, the excess release of destructive enzymes from dust- (or smoking-) stimulated inflammatory cells in association with a decrease in protective enzymes in the lungs.

65 Fed. Reg. at 79943 (Dec. 20, 2000). Consequently, Dr. Rasmussen's report is highly probative as he persuasively explains his finding of a coal dust induced respiratory impairment with the miner's work and smoking histories, physical symptoms, complaints, and objective medical data, including negative chest x-ray findings. Moreover, his opinion is based on premises regarding the development and characteristics of coal workers' pneumoconiosis that are consistent with the Department's position. Dr. Rasmussen's conclusions are further supported by the opinions of Drs. Buddington, Ducatman, and Buono.

C. Qualifications of the physicians

The physicians offering opinions at issue here are highly qualified. Dr. Buddington is board-certified in clinical and anatomical pathology. The board-certifications of Drs. Ducatman and Buono are not in the record, but they are professors of medicine at the University of West Virginia School of Medicine. Dr. Buono has a master's degree in public health and Dr. Ducatman has a master's degree in science. Drs. Castle, Zaldivar, and Rasmussen are board-certified in internal medicine and pulmonary diseases and they are NIOSH certified B-readers.

Of these physicians, Drs. Castle, Zaldivar, and Rasmussen possess board-certifications in the areas of medicine that are most useful in assessing whether the miner's lifetime respiratory impairment was due to coal dust exposure. Drs. Castle and Zaldivar have been authors of published works on medicine, they serve as clinical professors, and they hold significant pulmonary-related positions with area hospitals. However, Dr. Rasmussen has the most significant background in the study and treatment of coal dust induced lung diseases. He has been immersed in this area of medicine since at least 1969 when he received the American Public Health Association's Presidential Award for "exceptional service in the fight against black lung." He has been appointed to serve on several NIOSH and United Mine Workers of America committees addressing issues related to coal workers' pneumoconiosis. Dr. Rasmussen has also testified before both houses of the United States Congress as well as the West Virginia State Legislature on issues related to occupational pneumoconiosis. In sum, the focus of Dr. Rasmussen's long-term and distinguished medical career has been the study of occupational pneumoconiosis. For these reasons, it is determined that he is the most qualified physician offering an opinion in this record.

II

Weighing evidence together under *Compton*

In *Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000), the court held that, in order to establish pneumoconiosis, all evidence submitted under 20 C.F.R. § 718.202 (2004) must be weighed together. Specifically, the undersigned is required to compare chest x-ray findings under § 718.202(a)(1) with medical opinion findings under § 718.202(a)(4) to determine whether Claimant has sustained her burden.

In this particular claim, the undersigned found no clinical pneumoconiosis present as the preponderance of the chest x-ray evidence was negative for the disease. However, based on Dr.

Rasmussen's reports, as supported by the medical data and reports of Drs. Buddington, Ducatman, and Buono, the undersigned is persuaded that the miner suffered from legal coal workers' pneumoconiosis. After reviewing numerous conflicting x-ray interpretations of record, Dr. Rasmussen states that negative interpretations do not preclude a finding of legal coal workers' pneumoconiosis and he maintains that Dr. Zaldivar "ignored" a growing body of medical evidence that chronic obstructive pulmonary disease and emphysema can be caused by coal dust exposure, even in the absence of chest x-ray findings. He cites to a medical study in support of this opinion.

In its comments to the amended regulations, the Department stated that "NIOSH is the government agency charged with conducting research into occupationally-related health problems" and serves as the "statutory advisor to the black lung benefits program . . .." 65 Fed. Reg. 79939 and 79951 (Dec. 20, 2000). In this vein, the Department noted that NIOSH studies demonstrate that a diagnosis of chronic obstructive pulmonary disease "includes disease processes characterized by airway dysfunction: chronic bronchitis, emphysema and asthma." 65 Fed. Reg. 79939 (Dec. 20, 2000). Moreover, there is a body of scientific studies finding that "[d]eath from pneumoconiosis, chronic bronchitis, and emphysema has been related to cumulative dust exposure." 65 Fed. Reg. 79951 (Dec. 20, 2000).

While a determination of whether a miner's respiratory disease arose from his coal dust exposure must be determined in each claim based on expert medical evidence, it is incumbent upon the medical experts to provide well-reasoned, well-documented opinions. The miner had a 42 year history of coal mine employment and ceased working in 1985. He had a 35 to 45 pack year history of smoking cigarettes. The miner claimed to have quit smoking in 1986, but testing conducted during Dr. Zaldivar's and Dr. Crisalli's 1991 examinations indicate that the miner continued to smoke intermittently at that time. Both of these potential causative factors are extensive. Therefore, the undersigned is not persuaded by opinions based on a premise that simple coal workers' pneumoconiosis generally does not cause obstructive lung disease, emphysema, or chronic bronchitis. More explanation is required based on the specific circumstances in this case, particularly in light of the fact that the miner's respiratory impairment progressively worsened over time[6], he demonstrated consistent symptoms of wheezing, prolonged inspiratory or expiratory phases, and rhonchi, and he had such significant coal mining and tobacco abuse histories.

Based on the foregoing, the opinions of Drs. Castle and Zaldivar are less probative with regard to whether the miner suffered from legal pneumoconiosis to the extent they focused on negative chest x-rays and the obstructive nature of his impairment in rendering their opinions. They also premised their opinions on findings that coal dust exposure does not cause the development of centrilobular emphysema. Dr. Rasmussen's opinion, on the other hand, is based on grounds consistent with the Department's position, *i.e.* that coal dust exposure can cause

[6] Indeed, the miner's overall respiratory condition worsened significantly over time notwithstanding his response to bronchodilators due to the asthmatic component of his impairment. In this vein, Drs. Castle and Zaldivar noted that "improvement after bronchodilator (was) significant only in so far as the percentage but not as far as the absolute improvement."

obstructive lung disease, including centrilobular emphysema, and simple pneumoconiosis may progressively worsen even in the absence of continued exposure to coal dust.

Under the medical findings of this particular case, Dr. Rasmussen properly takes into account the miner's symptoms and testing results as well as the extensive work and smoking histories established on this record. Importantly, Dr. Rasmussen stresses that the miner was engaged in coal mine employment prior "to the institution of dust suppression in the coal mining industry . . .." In one of his reports, Dr. Rasmussen reasonably states:

> This patient's markedly reduced diffusing capacity and his abnormal gas exchange during exercise . . . coupled with the marked reduction in diffusing capacity clearly indicates that this patient has pulmonary parenchymal destruction. Coupling this with his severe ventilatory impairment would lead one to believe that he has pulmonary emphysema. Interestingly, however, he has no increase in total lung capacity suggesting that he has at least a significant component of pulmonary fibrosis. Both emphysema and fibrosis are consistent with occupational pneumoconiosis.

Dr. Rasmussen's finding that legal coal workers' pneumoconiosis, *i.e.* coal dust related chronic obstructive lung disease and emphysema, contributed to the miner's respiratory deterioration is consistent with his progressively worsening and irreversible ventilatory findings as well as the miner's consistent symptoms of wheezing, rhonchi, and prolonged inspiratory or expiratory phases across examinations. Also, as noted by Dr. Rasmussen, it is consistent with the miner's reduced diffusing capacity. Based on the foregoing, preponderantly negative chest x-ray findings do not detract from findings of legal pneumoconiosis.

Notably, the 1991 examination of Dr. Zaldivar demonstrated a continuing, significant smoking history. However, an examination conducted by Dr. Crissali earlier in the same month produced results that were "borderline" between a smoker and non-smoker as noted by Dr. Zaldivar. Thus, while the miner had an ongoing smoking habit, it was intermittent. Consequently, Dr. Rasmussen's opinion remains probative. He persuasively opines that, in addition to coal dust exposure, smoking also contributed to the miner's respiratory ailment. This is consistent with findings of gas trapping and an elevated CO2, or hypercarbia, as noted by Drs. Zaldivar and Castle.

On balance, after weighing all of the evidence under 20 C.F.R. § 718.202(a)(1) and (a)(4) (2004), Claimant has established that the miner suffered from legal coal workers' pneumoconiosis based on Dr. Rasmussen's well-reasoned, well-documented medical opinions. His findings of legal coal workers' pneumoconiosis are not diminished by the preponderantly negative chest x-ray evidence of record.

III

Cause of death

Of the physicians who addressed the cause of the miner's death, the Board held that Administrative Law Judge Holmes properly credited the opinions of Drs. Ducatman, Buono, and Rasmussen that "smoking and coal dust exposure led to the miner's total respiratory failure." Further, Dr. Hynes, one of the miner's treating physicians, concluded that the miner died due to "black lung."

Drs. Fino, Castle, and Endres-Bercher failed to diagnose the presence of clinical or legal coal workers' pneumoconiosis. Consequently, their opinions are less probative in determining the cause of the miner's death. *Scott v. Mason Coal Co.*, 289 F.3d 263 (4th Cir. 2002) (less weight may be accorded to physicians' opinions premised on a finding of no pneumoconiosis that is contrary to the ALJ's findings).

Drs. Zaldivar and Morgan also failed to diagnose the presence of clinical or legal coal workers' pneumoconiosis. However, they stated that, even if the miner suffered from the disease, his death was due solely to smoking-induced obstructive lung disease. Specifically, Dr. Zaldivar stated the following:

> Mr. Price had bullae radiographically. Bullae due to emphysema is never a manifestation of coal workers' pneumoconiosis. It is a manifestation of emphysema which causes centrilobular and, in the case of bullae, panacinar emphysema.

Similarly, Dr. Morgan stated that the miner's respiratory impairment was related to emphysema and small airways disease and that any minor presence of coal workers' pneumoconiosis would "not affect his lung function." He reiterated that there was no evidence that the miner suffered from coal workers' pneumoconiosis "based on radiographic examination." Dr. Morgan did conclude, however, that the miner's death was due to severe chronic airflow limitation (emphysema and chronic bronchitis) due to cigarette smoking.

Even though Drs. Morgan and Zaldivar did not diagnose the presence of pneumoconiosis, they stated that, even if the disease was established, their opinions as to the cause of the miner's death would not change. Their reports are not well-reasoned and carry little probative value. In *Soubik v. Director, OWCP*, 366 F.3d 226 (3rd Cir. 2004), the court held that a physician's failure to diagnose pneumoconiosis would have an adverse effect on his ability to assess whether the miner's death was due to the disease:

> Common sense suggests that it is unusually exceedingly difficult for a doctor to properly assess the contribution, if any, of pneumoconiosis to a miner's death if he/she does not believe it was present.

Similarly, in this case, Drs. Zaldivar and Morgan did not believe that the disease was present. As a result, they offered only conclusory statements regarding the cause of the miner's death in the event that presence of pneumoconiosis was assumed. As a result, their opinions are not well-reasoned or well-documented in support of a finding that coal workers' pneumoconiosis did not contribute to the miner's death.

Consequently, a preponderance of the evidence supports a finding that the miner suffered from legal coal workers' pneumoconiosis and as previously noted by Administrative Law Judge Holmes and affirmed by the Board, based on the opinions of Drs. Ducatman, Buono, and Rasmussen, this impairment hastened his death. 20 C.F.R. § 718.205 (2005).

As a result, a preponderance of the evidence supports a finding that the miner suffered from legal coal workers' pneumoconiosis and this impairment hastened his death. 20 C.F.R. § 718.205 (2004).

ORDER

IT IS ORDERED that Employer shall pay to Claimant, Ola Mae Price, all benefits to which she is entitled commencing as of January 1997, the month in which the miner died; and

IT IS FURTHER ORDERED that, within 30 days of the date of this *Decision*, Claimant's counsel shall file, with this Office and with opposing counsel, a petition for a representatives' fees and costs in accordance with the regulatory requirements set forth at 20 C.F.R. § 725.366 (2004). Counsel for the Director and for Employer shall file any objections with this Office and with Claimant's counsel within 60 days of the date of this *Decision*. It is requested that the petition for services and costs clearly state (1) counsel's hourly rate and supporting argument or documentation therefor, (2) a clear itemization of the complexity and type of services rendered, and (3) that the petition contains a request for payment for services rendered and costs incurred before this Office only as the undersigned does not have authority to adjudicate fee petitions for work performed before the district director or appellate tribunals. *Ilkewicz v. Director, OWCP*, 4 B.L.R. 1-400 (1982).

A

Thomas M. Burke
Associate Chief Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the

administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is:

Benefits Review Board
U.S. Department of Labor
P.O. Box 37601
Washington, DC 20013-7601

Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed. At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481. If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).